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# Medicine for the Layman

## Obesity and Energy Metabolism

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## Foreword

Americans continue to demand a greater role in deciding issues that affect their health. Increased health awareness and the convincing evidence linking life style, risk factors, and specific diseases have accelerated our need to know.

The Clinical Center, recognizing the importance of providing information to facilitate intelligent decisions on health issues, created a unique lecture series featuring distinguished physician scientists working at the frontiers of biomedical research at the National Institutes of Health.

The series — Medicine for the Layman — has provided an opportunity for thousands of people to learn more about how their bodies work and what they can do to maintain or improve their health.

This publication is one of several adapted from the series. It is our sincere hope that you will find this material interesting and enlightening.

John L. Decker, M.D.
Director, The Clinical Center



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## Obesity and Energy Metabolism

Presented by
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Dr. Salans is a noted diabetologist and expert on obesity and nutrition. His research has centered on the relationship of obesity to diabetes, and particularly on the cellular and metabolic character of the adipose tissue (body fat) in these disorders. Although all obesity is defined as an increase in body fat, two kinds are known to exist. Some obese people have an increased number of fat cells, whereas others have normal numbers of very large fat cells. Increased fat cell size, however, characterizes all forms of obesity. Dr. Salans' studies suggest that these cellular patterns may have an important bearing upon the medical consequences of obesity, such as the abnormalities of insulin secretion and action, and glucose and lipid metabolism in diabetes, as well as other endocrine and metabolic implications. He has published more than 50 scientific papers on these subjects and has received honors and awards that include the National Institutes of Health Director's Award.

Dr. Salans came to NIH in 1976 as Associate Director for Diabetes, Endocrinology, and Metabolic Diseases, National Institute of Arthritis, Diabetes, and Digestive and Kidney Diseases (NIADDK). He also served from 1977 to 1982 as Chief, Cellular Metabolism and Obesity Laboratory, NIADDK.

Prior to coming to NIH, Dr. Salans was Associate Professor of Medicine and Head of the Section of Endocrinology and Metabolism. Previously, he was at the Rockefeller University and, from 1974 to 1975, he was an invited professor at the Institut de Biochimie Clinique, University of Geneva, Switzerland.

Dr. Salans is a member of several professional societies, including the American Society of Clinical Investigation, the American Diabetes Association, the Endocrine Society, and the American College of Nutrition.

A native of Chicago, Dr. Salans earned his B.S. degree at the University of Michigan, and in 1961 he received his M.D. degree with honors at the University of Illinois College of Medicine.

## Obesity and Energy Metabolism

Obesity is one of the most written about, most discussed, and least understood public health problems facing our nation. An enormous amount of misinformation, myth, and unsuccessful medical management surrounds this serious medical problem.

## Causes of Obesity

At the simplest level obesity is the result of eating more calories in food than required by the body. Many potential causes for this exist, and therefore physicians think of this disorder not as a single disease but as several different conditions with the symptom of overweight in common. In most cases the underlying cause of obesity in a given individual is unknown. Physicians are able to identify the specific cause in less than 5 percent of the people who suffer from obesity.

Obesity is a common problem varying in degree from mild to severe. Although the exact prevalence of obesity in the population is not known, one estimate suggests that about 30 million Americans are obese. In addition, there is evidence that the incidence of obesity may be increasing. A recent survey, for example, indicates that the body weight of adult Americans from 1970 through 1974 was considerably higher than the body weight of adult Americans from 1960 through 1962.

Several factors appear to influence the prevalence of obesity in the American population. While excess body weight can affect people of all

ages, obesity increases in prevalence with increasing age, peaking in middle age. Obesity is common throughout all segments of our society, but it appears to have a particular relationship to socioeconomic status. The lower the socioeconomic status, the greater the prevalence of obesity. Similarly, as the level of education decreases, the prevalence of obesity increases. Obesity among blacks in this country, particularly among black women, appears to be greater in proportion to population than obesity among whites. These socioeconomic trends, however, do not apply to individuals at or below the poverty level; these individuals have a low incidence of obesity.

Genetic factors appear to be important in obesity although their exact nature or role is not well understood at present. Obesity tends to run in families, a fact which reflects, at least in part, the genetic or hereditary basis for obesity. But, environmental, lifestyle factors such as attitudes and practices regarding eating, and physical activity also are important determinants of obesity. Thus, it is probable that both genetic and environmental factors contribute to the development and perpetuation of obesity, and it is often very difficult to separate these two factors in a given individual.

## Health Consequences of Obesity

Obesity is associated with serious and significant negative health consequences. For example, obesity appears to be associated with serious consequences for the quality

and length of life.

In adults the mortality rate for obese individuals exceeds the expected death rate for other individuals in that age group, especially for individuals who are more than 30 percent above standard body weight as defined by the Metropolitan Life Insurance Company's 1959 height-weight table. Individuals who are overweight by less than 30 percent are also at increased risk for mortality. Thus, a person does not need to be massively overweight to face an increased risk of death; even a moderate or mild degree of overweight can increase the risk of mortality at each age group. In large part, this appears to be related to the fact that the obese individual is prone to a greater variety of serious and lifethreatening diseases than is the non-obese person.

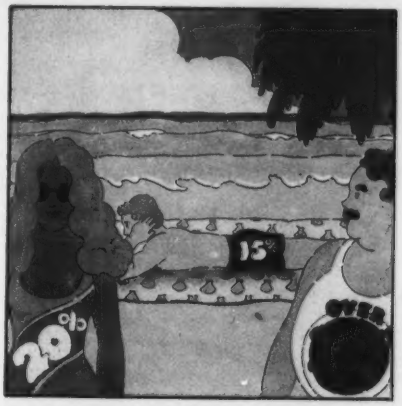
The obese individual runs a greater risk of developing diabetes mellitus than the non-obese person. The chance of developing diabetes may be two or three times greater for the obese person. The health consequences of diabetes are so serious and widespread that public health experts view the increase in obesity with alarm.

Researchers do not fully understand the mechanism whereby obesity increases the risk of developing diabetes. Information from several laboratories, in this country and abroad, suggests that obesity somehow interferes with the action of insulin. In addition, in some obese individuals there appears to be a decreased ability of the pancreas to secrete sufficient amounts of insulin into the blood. As a consequence of insufficient insulin ac-

tion the body's regulation of glucose (sugar) metabolism may be abnormal and the blood glucose level may become elevated. A blood test, urinalysis, or glucose tolerance test can detect excessive amounts of glucose in the blood.

If the obese individual with diabetes and an abnormal glucose metabolism loses weight by losing body fat, insulin's ability to regulate glucose metabolism improves, the blood insulin level returns to or towards normal and the blood glucose concentration decreases. Moreover, the symptoms of diabetes often disappear.

The obese individual also runs an increased risk of developing a variety of cardiac diseases. The most serious of these conditions, atherosclerotic heart disease, can lead to heart attacks. Although the reason for the increased risk of heart disease associated with obesity is not known with certainty, researchers believe that one explanation is that obesity tends to raise blood fat levels. The obese individual is at substantially greater risk of having higher levels of triglyceride (a molecule carrying three fatty acids) and cholesterol in the blood than the non-obese individual. Both triglyceride and cholesterol have been implicated as factors in causing heart attacks. Obesity, especially when associated with diabetes, may also lower the level of a particular lipoprotein that appears to be protective against coronary heart disease. With weight loss and reduction of body fat the levels of triglyceride and of cholesterol in the blood decrease.



Additional factors probably contribute to the relationship between obesity and heart disease. The obese person is, for example, predisposed to high blood pressure. Although not a disease in itself, high blood pressure contributes to vascular disease and degenerative diseases of the liver and kidney. As with diabetes and plasma fats, blood pressure usually improves with loss of weight and body fat.

Obesity affects the individual in many other ways. It places severe stress on the internal organs, the musculoskeletal system, the digestive tract, and the psychological state of the obese person.

Obese individuals often find that they are outcasts in our society. They may encounter discrimination in employment as well as daily living situations. This treatment generates emotional stress, an important health consequence of obesity.

## What is Obesity?

Obesity is an excessive increase in body fat. In man and in animals, fat in the body is stored primarily in specific cells (adipose or fat cells) in the adipose (fat) tissue.

Microscopic examination shows that a fat cell is very different in appearance from other cells. Most cells contain a large amount of cytoplasm, with the cell nucleus near the center of the cell. Fat constitutes almost the entire area of the adipose cell, and the cytoplasm and nucleus are displaced. Thus, fat, in the form of triglyceride, is the major component of the adipose cell.

In the non-obese adult male approximately 10 to 15 percent of the total body weight is fat. In the non-obese woman, fat may constitute up to 20 percent of total body weight. If fat comprises more than 20 percent of the total body weight, the individual is obese.

At the present time there is no practical or simple way to determine the amount of a person's body weight which is fat. This measurement cannot be determined by a physician in his office with any accuracy. Only a sophisticated laboratory has equipment capable of measuring the fat/body weight ratio. For this reason, people have developed alternative ways to assess obesity. The most common index of obesity is body weight. Several "standard tables" exist which provide recommended body weight in relation to height and body frame. The body weights recommended in these tables are those associated with the longest lifespan in a population of insured individuals. In 1983, the Metropolitan Life Insurance Company introduced new height-weight tables in which the weight ranges were shifted upward. Most researchers believe that the body weights recommended in the new 1983 tables are too high at each height and that the body weights recommended in the original 1959 Metropolitan tables are preferable.

If an individual weighs more than the "ideal" or "average" weight for his or her age, height, and body frame, as shown in the "standard" table, he or she is overweight. An individual who is more than 15 to 20 percent overweight is considered to be definitely obese. Obesity may also, and probably does, exist in individuals who are 5 to 15 percent over the

"ideal" or "average" body weights shown in the "standard" tables.

There are difficulties, however, in determining obesity from the "standard" weight/height tables. For example, of two men with the same weight and height, one may be obese, and the other not. Both may weigh 225 pounds and be six feet tall; one man is an athlete and his weight is due to increased muscle mass, while the weight of the second man, in poor physical condition, is due to an increased amount of fat. Both of these men would be classified as overweight according to the "standard" tables, but only one is obese, because obesity is an increase in body fat and not an increase in muscle.

There are other means of indirectly assessing body fat, such as measurement of skin fold thickness. While these are simple techniques which can be utilized by physicians, they are of limited accuracy and usefulness. It is clear, therefore, that new methods must be developed which will enable physicians to determine body fat easily and reliably.

## Adipose Tissue and Energy

In man the adipose tissue (fat) has a very important function; it serves as the principal energy storage organ of the body. Energy is measured in calories. Adipose tissue is the caloric reservoir of the body.

Chemical energy — calories — originates in food ingested as nourishment. The digestive system enables the body to extract a portion of the chemical energy in the food, and to use the calories for fuel. Only a portion of this energy, however, can be used by the body. Re-

searchers estimate that the body uses roughly 40 percent of the potential chemical energy in foodstuffs as fuel for cells. The remainder is converted to heat and is lost from the body.

Foodstuffs are composed of carbohydrates, fats, and proteins; energy is extracted from all three nutrients. The nutrient reacts with oxygen, and with the assistance of enzymes to facilitate the process, releases energy. The energy extracted is converted to adenosine triphosphate, ATP, a molecular compound with three phosphate groups. The phosphate bonds are high energy bonds, so that when a phosphate group is split from ATP the energy is released for the body's

This energy is used by the body for two purposes. First, it supports vital bodily functions such as respiration, the action of the heart muscle, synthesis and secretion of compounds, and maintenance and repair of tissues. These and other vital bodily functions require energy needs, the same as an automobile requires gasoline. A sufficient amount of energy (number of calories) must be supplied to the body for it to perform its vital functions; this energy is therefore essential for life. When the energy or calories released from ATP which enter the cells of these tissues and organ systems are sufficient to meet the cells' energy needs, the excess calories are stored as fat in adipose cells. Generally, fat is stored as triglyceride in the adipose tissue for future use.

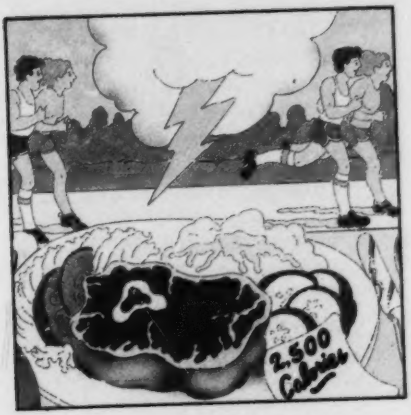
Secondly, energy is used by the body to perform physical activity. Performance of a given amount of activity requires that a certain amount of energy be supplied to the

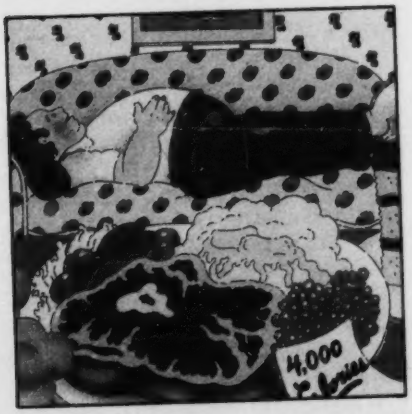
body. When the energy or calories released from ATP which enter the tissues performing this physical activity is sufficient to meet the need, the excess calories are stored as fat.

When the amount of energy provided by the calories in the ingested food is equal to the amount of energy needed by the body to perform all of its vital functions plus any additional physical activity, the individual is said to be in energy balance. In this situation the person will maintain the amount of adipose tissue and body weight at a constant level. When the amount of energy provided by the calories in the ingested food is less than the amount of energy needed by the body, the individual is said to be in negative energy balance. In this situation the adipose tissue will be broken down to provide the energy needed by the body, this tissue will decrease in size and the individual will lose body weight. When energy intake in food is greater than the amount needed by the body, the person will increase the amount of adipose tissue, that is, will become fatter, and will increase in weight.

In an obese person the caloric intake is consistently greater than the amount the body needs to maintain its vital functions and to perform daily activities. The excess calories go to fat. Since the individual is in positive energy balance, the body weight increases and body fat increases, expanding the adipose tissue.

At rest, an adult man requires about 1200 to 1500 calories a day to maintain his vital functions. If the person is doing something other than lying at rest, such as working or walking, his energy requirements will rise. In order to maintain his



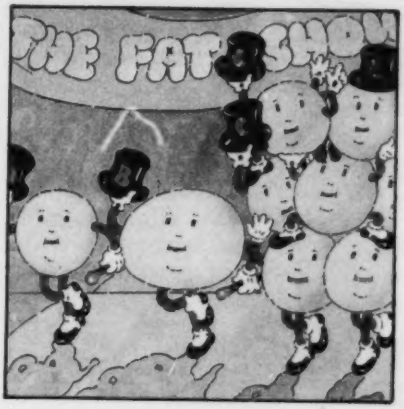

body weight and energy balance, he will have to ingest perhaps 2000 calories a day. If he does, his body weight remains stable, whereas if he ingests less, his body weight decreases. If he ingests more calories than he expends in his activities, the excess calories are converted to triglyceride and stored in the adipose tissue. Vigorous physical activity, such as exercise, will raise energy requirements even more and caloric requirements will increase. The same principles governing energy balance and body weight just described still apply, however.

The same principles apply to women, although the energy requirements of men and women do differ slightly. A woman at rest requires approximately 1000 calories to 1300 calories a day, on the average. When she is active she will require more calories to maintain her weight. Of course, if she ingests more than she needs she will store the excess in adipose tissue and will gain fat and weight.

Energy requirements at rest and during activity will vary from person to person. People have different rates of basal metabolism; therefore people have slightly different caloric needs. In addition, some peoples' bodies are physiologically more efficient than others at extracting and utilizing the energy within foodstuffs. One must tailor a nutritional assessment and caloric intake plan to each person individually. Although there is at present no definitive or conclusive data to support the view that subtle differences in the ability to extract or utilize calories cause obesity, recent research may suggest that such differences might contribute to the

development or perpetuation of the obese state in some individuals. On the other hand there is a real need for continued research on whether such metabolic differences between individuals can be causative factors in obesity.

One of the paradoxes of obesity is that a larger individual does require more energy to maintain bodily functions in the resting state. The obese person's body must work harder to move his chest wall in and out during respiration, or to pump blood to the large mass of tissue. These actions cost the obese person more energy. Moreover, the energy cost of physical activity in the obese individual is greater than in the non-obese person. Thus, if an obese and non-obese individual are performing identical physical activity and are eating the number of calories required to maintain constant body weight in the non-obese person, the obese individual would be eating fewer calories than his body needs and should lose weight. Unfortunately, the obese individual usually doesn't eat the same number of calories as the non-obese. Moreover, when a person gains an excessive amount of weight, it becomes more difficult to be physically active. With a decrease in activity, energy requirements are reduced, and the individual gains weight more easily, compounding an already difficult problem. Thus, an increase in food intake and a decrease in physical activity contribute to obesity. Research supports the conclusion that overeating is the major cause and that a decrease in physical activity is a contributing factor in the creation of positive energy balance and the



resultant expansion of the adipose tissue and development of obesity.

## How Does Adipose Tissue Grow?

Recent studies indicate that the adipose tissue expands in obesity in two ways. Storing more fat within the adipose cells already in the body is one way of increasing the adipose tissue mass. A second way that the body can expand its adipose tissue is to make more adipose cells and fill them with fat.

There are two types of obesity based on the two ways the body can increase the mass of fat tissue. In *hypertrophic* obesity, the individual has the same number of fat cells as the non-obese person, but the fat cells are much larger. In *hyperplastic hypertrophic* obesity, the obese person has not only enlarged fat cells but also a much greater number of fat cells. In general, there appears to be a good correlation between the degree of overweight and the number of fat cells in the body: the greater the obesity the more likely it is to be associated with adipose hypercellularity.

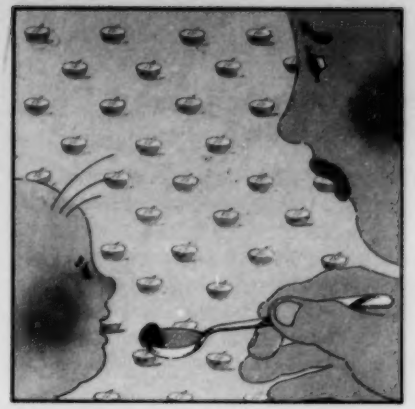
The ability to measure and to study fat cell size and number has led researchers to two important conclusions regarding obesity. First, hyperplastic hypertrophic obesity appears to be irreversible. Once the number of fat cells in someone's body has been increased, it remains elevated thereafter, even if the person loses weight. The results of many studies are illustrated by the following example. An extremely obese man is brought into the hospital where fat cell size and total number of fat cells in his body are

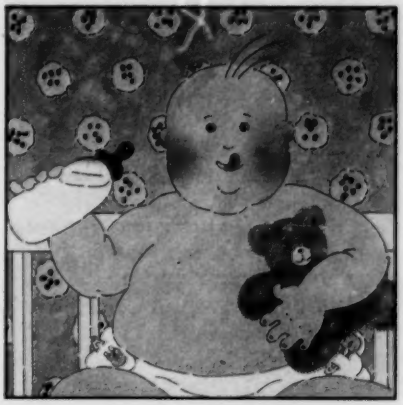
measured. The individual is found to have large fat cells in addition to many more fat cells than normal. After many months of caloric restriction the man has achieved his normal body weight and is studied again. He is found still to have an abnormally increased number of fat cells. His fat cells themselves have decreased in size, but his hypercellularity remains.

A second conclusion is that the number of fat cells in the body can be increased by overeating. This can occur at any age—infancy, childhood, adolescence, or adulthood, but early life seems to be an especially sensitive period for adipose cell proliferation. For these reasons, physicians and investigators are focusing on the preventive aspects of obesity.

Overfeeding in the earliest days of life can lead to an abnormal increase in number of fat cells and the irreversible condition of hyperplastic hypertrophic obesity in experimental laboratory animals. The same relationship has not yet been shown in man. However, if overnutrition early in the life of man can be shown to lead to obesity in adult life, perhaps irreversible obesity can be prevented by elimination of the practice of overfeeding infants.

Hypercellularity of the adipose tissue has also been demonstrated to have its onset in adult life particularly in those individuals who are ingesting especially large amounts of excess over prolonged periods of time. This adipose hypercellularity of adult onset also appears to be irreversible. Clearly then, attention needs to be focused on preventing overeating during





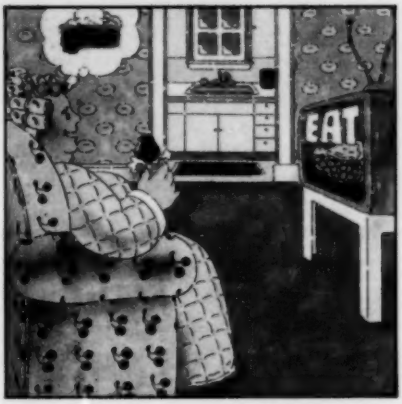
adult life.

The purely hypertrophic type of obesity appears to be more reversible than the hyperplastic hypertrophic form. This type of obesity occurs, most often, in individuals who are not massively obese and were not overweight in infancy and childhood, but who become obese as adults. Overeating and underexercising are responsible for hypertrophic obesity. The cause of overeating is still unknown.

## Opportunities for Obesity Research

The implications of these and other research findings for obesity are important to consider. First, scientists recognize that there is a great deal more to be learned about obesity. The fundamental processes which control energy balance, eating behavior, and efficiency of energy utilization require more study. Basic and clinical research in these areas continues and researchers are optimistic about the progress of their studies.

Secondly, the knowledge gained may provide several important implications for the prevention of obesity. Researchers have demonstrated the potential harmful effects of overfeeding infants. Often cultural attitudes, taught by generations of parents to their children, are erroneous. Consider these common directives: "You must clean your plate" or "the healthy baby is the fat baby." Without interference, a child will stop eating when he or she is no longer hungry. With forced feeding the desire to please mother may subvert the body's normal physio-



logical message of satiation. In part, obesity in later life may be caused by this confusion between eating and "being good."

Overeating seems to be endemic to the American adult population. Our society is oriented towards eating. Food advertisements are persuasive on television. Fast food restaurants and automats are everywhere; buildings contain canteens, often on every floor. Although it will be extremely difficult to effect, Americans will have to change their life style—their beliefs, attitudes, and practices regarding eating and exercise—if we are to solve the problem of obesity.

Lack of exercise, the other major factor in adult obesity, applies to most men and women in our society. American adults tend to participate in physical activities as spectators rather than as participants. Again, only a fundamental change in life style can alter the pattern. The jogging and running movement may indicate a change for the better.

Although it is difficult to alter social attitudes, public education is our only effective weapon. The school system is a place to begin. Children should be more effectively taught the importance of regular physical activity to good health. Teachers must teach proper nutrition in classes. Cafeterias have the responsibility to put these lessons into practice. At the present time school lunches do not set a good example of proper eating habits for the students.

Good habits begin in the home. We must educate our children when they are very young about the value of nutrition. We must teach them how to select foods from the basic food groups to avoid overeating. Parents, health educators, physicians and others must direct their efforts towards implementation of nutritional education. By focusing on childhood, where lifetime patterns are established, we may someday be able to prevent some forms of obesity.

**Q.** If they are put on the same diet, are people with hypertrophic obesity and those with hyperplastic hypertrophic obesity likely to lose weight at the same rate?

**A.** Clinical experiments have shown that people with both types of obesity will lose weight at the same rate. The ability to keep the weight off, however, differs. All obese individuals who lost weight in a controlled environment have a great tendency to regain that weight once they return to their former home environment. Individuals with simple hypertrophic obesity, who have a normal number of fat cells, are, however, more successful at keeping the weight off once they return home.

Researchers do not know why one type of obesity is more responsive to keeping weight off. One hypothesis is that the *appestat*, the mechanism in the brain which controls hunger and satiety, might be related to the number of fat cells in the body. Perhaps the signal the appestat receives is stronger than it should be in people with hyperplastic obesity.

**Q.** Are there people with an excess number of fat cells, but who are not overweight?

**A.** There may be some normal weight individuals with hypercellularity but it would be a very small percentage of the population. Most who fall in this category are people who were obese in the past and have lost weight.

**Q.** What effect do hormones and the endocrine systems have on obesity?

**A.** The endocrine system governs the hormones which control the actions of all organs. In an extremely limited number of patients obesity

can be caused by an endocrine dysfunction. For example, a patient who does not have enough thyroid hormone is often overweight. The obesity of hypothyroidism is generally mild, however, and if treated, the individual with obesity due solely to hypothyroidism will lose weight and become normal in body weight. Hypothyroidism, however, is an extremely rare cause of obesity and in the overwhelming majority of cases obese patients are not hypothyroid. The taking of thyroid hormone in almost all obese patients reduces their finances, not their weight.

An excess of adrenocorticosteroids, called "Cushing's syndrome," is related to obesity, but again, this condition is a rare cause of obesity. The hypothalamus is one endocrine gland which may have a role in development of obesity. The hypothalamus may cause overeating or a metabolic effect which increases fat synthesis and fat storage. For example, animal experiments have demonstrated that destruction of certain parts of the hypothalamus causes overeating and obesity by causing excessive secretion of insulin. Insulin is a hormone which stimulates fat synthesis, facilitating storage of fat.

Physicians have observed that men with tumors of the hypothalamic region become obese. Thus endocrine dysfunction as a cause of obesity remains a possibility in a small number of instances.

At the same time, it is clear that a variety of hormones and hormone-like substances influence metabolism and may also affect eating behavior. In the past few years researchers have identified hormones and peptides in the intestine and the brain which appear to be either identical or similar and which may play an important role in regulating and integrating eating behavior and digestion.

**Q.** What kind of obesity is involved in endocrine disorders?

**A.** Research findings suggest that the rare cases of obesity caused by hormonal imbalance, are hypertrophic in character; there is no increase in cell number.

**Q.** How does insulin affect one's ability to transform chemical energy into mechanical energy?

**A.** Insulin is a hormone that is synthesized in the beta cells of the pancreas. It circulates in the blood and acts upon a variety of cells, including muscle, fat tissue and the liver. Insulin allows the glucose in the bloodstream to be used inside the cell. If the cell receives amounts of glucose in excess of its needs, the glucose is converted to fat and stored. Insulin also affects fat and protein metabolism. Insulin stimulates fat synthesis and inhibits fat breakdown, although researchers do not understand the method by which this action occurs. Insulin also facilitates the transport of amino acids, the building blocks of protein, into the cell, and prevents the protein in the cell from breaking down.

Scientists do not know exactly how insulin exerts its effects on cells, but it appears to have three major actions: binding to specific insulin receptors on the cell surface, stimulation of nutrient transport into the cell, and stimulation of nutrient metabolism inside the cell. In some

obese patients insulin does not interact with cells in the muscle, fat, and liver in a way which causes the proper response. Alterations in insulin's interaction with the receptors of these cells, in its effects on the transport processes or on the cells metabolism, may cause this lack of response.

Insulin's stimulation of the synthesis of protein and fat and of the storage of glucose as glycogen provides the cell with several potential sources of the energy necessary for its vital functions. ATP is generated from the metabolism of these substances and cells obtain their energy from the breakdown of ATP. If insulin fails to mediate these processes, the cells' source of energy is diminished.

In the obese person there seems to be an impairment in the ability of insulin to regulate these metabolic processes in a normal manner. One consequence is that glucose does not enter and leave the blood properly and its concentration tends to rise in the blood. That is the connection between obesity and diabetes. On the other hand, even though this problem exists, the obese individual is able to get calories into the cells and store this energy as triglyceride. That is obvious since if such compensation did not occur a state of negative energy balance would exist, adipose tissue and body weight would decrease and there would be no obesity.

Scientists do not yet fully understand the mechanism of this compensation, but it probably reflects the fact that the obese are consuming so many more calories than needed and the body has available energy rich nutrients other than glucose, such as fat.

**Q.** How do pre-digested liquid protein diets work and why are they so dangerous?

**A.** Pre-digested protein diets work for the following reason: individuals following the directions on the label are ingesting fewer calories than their bodies utilize. People on the diet maintain a negative caloric balance, so they lose body weight and body fat. All diets work on the same principle: eat fewer calories than your body needs. Thus, diets that suggest you eat all you want, high-fat diets for example, really only work when the individual actually ingests fewer calories than the body needs. Generally, after a few days the diet becomes so unpalatable that the desire for foods diminishes. Once the dieter eats fewer calories than utilized by the body, negative caloric balance is established and weight loss ensues.

Liquid protein diets taken in the prescribed amounts are very low in calories. Their ingestion leads to a state of negative energy balance or semi-starvation. A variety of biochemical changes occur in addition to negative energy balance during starvation. Starvation depletes the body of vital minerals and trace elements; blood potassium level, for example, tends to fall.

Potassium is an important element in the maintenance of normal cell function. The heart muscle is especially sensitive to the level of potassium in the blood. Patients on the liquid protein diets have been reported to have abnormalities in potassium as well as other minerals. It has been suggested, although it

remains to be proven, that the severe heart problems experienced by some women on the liquid protein diet are related to their depleted potassium levels.

Another effect of starvation is a rise in uric acid levels in the blood. People on this diet have an increased tendency to develop gout and kidney stones. A physician must be aware of these potential dangers and follow the patient carefully. By monitoring for these changes through laboratory tests and physical examination, the physician can correct them at their earliest appearance.

Because of the unknown and possibly lasting effects of any "quick weight loss" or fad diet, including the liquid protein diet, health experts do not recommend them except under certain experimental conditions. A healthy reducing diet should consist of foods from the four basic food groups, including fat, protein, and carbohydrate. A qualified physician or nutritionist should formulate the diet plan specific to the individual's needs; every person is different.

**Q.** We have heard about the effect of over-feeding the newborn infant, but what about infant malnutrition? What are its consequences in later life, and which is more important?

**A.** Certainly one should not starve or undernourish infants or children. Undernourishment has even more dire consequences than overfeeding. The idea is to achieve a caloric intake that is optimal for health, producing neither under- nor over-nutrition. What constitutes an optimal diet for an infant is, however, unknown at this time. The general impression is that what many have considered "optimal" intake in the past may actually represent over-nutrition. This is an area that requires continued research. Certainly, malnutrition of any type is to be avoided.

**Q.** Do you believe that mass education, making the public more aware of the risks of obesity, overeating and the lack of exercise, can counterbalance Madison Avenue's desire to sell the "good life."

**A.** We must recruit all segments of the society to the cause of public health. If advertisers will join in the effort of educating the public, I think they will find many new markets. But they must change the focus of their campaigns from those currently too often practiced which misinform and mislead the public. Evidence suggests that education can work. Consider what has happened with coronary heart disease and hypertension. The program of public information dissemination and education has affected prevention, detection and treatment of these problems. We must not forget that the Madison Avenue people also can be educated. With their help, we may be able to make progress in the area of obesity.

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